Amendments to the Specification:

Please replace the paragraph (or section) beginning at page 1, line 4, with the following redlined paragraph (or section):

The present application is a divisional of U.S. Application No. 09/952,768, which application is a continuation of U.S. Application No. 09/291,692, filed April 13, 1999; which application is a continuation of ~~pending~~ United States Patent Application No. 08/882,429, filed June 25, 1997, now abandoned; which application is a continuation of United States Patent Application No. 08/665,220, filed June 14, 1996, now issued as United States Patent No. 5,786,173; which application is a continuation-in-part of United States Patent Application No. 08/618,408, filed March 19, 1996, now issued as United States Patent No. 5,851,815; all prior applications are hereby incorporated herein by reference.

Please replace the paragraph (or section) beginning at page 5, line 3, with the following redlined paragraph (or section):

Figure 3 shows the amino acid sequences of Mch4 and Mch5 and their homology to other ASCP sequences. (A) Colinear alignment of human FADD (SEQ ID NO:64) with the two FADD-like domains in both Mchk4 and Mch5. These domains have been denoted as Mch4A (amino acid residues 18-105; SEQ ID NO:65), Mch4B (amino acid 112-189; SEQ ID NO:66), Mch5A (amino acid 4-77; SEQ ID NO:67) and Mch5B (amino acid 128-205; SEQ ID NO:68). The first domain of Mch4 (Mch4A) has the highest homology to the N-terminal 79 amino acid long FADD (gb accession #U24231) death effector domain (37% identity, 57% similarity) than the second domain (Mch4B) 22% identity, 53% similarity). Mch4A also shows high homology to PEA-15 (gb accession #X86694) and KIAA0179 (gb accession #D80001) proteins. Mch4B shows some homology to SRB7 (gb accession #U46837) and yeast cdc4 (gb accession #Z45255) proteins. (B) Multiple sequence alignment of all known human ASCPs (*i.e.*, Mch5 (SEQ ID NOs: 12-16), Mch4 (SEQ ID NOs: 17-21), Mch3 (SEQ ID NOS: 22-26), Mch2 (SEQ ID NOs: 27-31), CPP32 (SEQ ID NOs: 32-36), ICE (SEQ ID NOs: 42-46), TX (SEQ ID NOs: 47-51), ICEre1IIII (SEQ ID NOs: 52-56), ICH-1 (SEQ ID NOs: 57-61) and the nematode Ced-3 ASCP (SEQ ID NOs: 37-41). The active site pentapeptide QACRG/QACQG (SEQ ID NOs: 11 and 10 respectively) is boxed. Based on crystal structure of ICE, the numbered residues

within the ICE sequence are involved in catalysis (open boxes), and binding the substrate-carboxylate of P1 Asp (open circles). The residue adjacent to the substrate P2-P4 amino acids are indicated by closed triangles. D/X indicates known and potential processing sites between the small and large subunits of ASCPs. The Roman numbers on the right indicate the three ASCP-subfamilies; the Ced-like subfamily (I), the ICE-like subfamily (II) and the NEdd2/Ich-1 subfamily (III). The asterisk indicates the nonconservative Arg to Gln substitution in Mch4 and Mch5.

Please replace the paragraph (or section) beginning at page 27, line 17, with the following redlined paragraph (or section):

As shown above in Table I, the K_m values of Mch4 for the two peptide substrates DEVD-AMC (SEQ ID NO: 71) and YVAD-AMC (SEQ ID NO: 72) are similar. These values contrast with those for CPP32, where the K_M for the YVAD-AMC (SEQ ID NO: 72) substrate is >35-fold higher than the K_M for the DEVD-AMC (SEQ ID NO: 71) substrate (Fernandes-Alnemri et al. supra (1995b)). These kinetic references are further illustrated by the ratio of Vmax/Km for the DEVD-AMC (SEQ ID NO: 71) substrate. Specifically, CPP32 possesses a >500-fold higher specificity for this substrate compared to Mch4 ($V_{max}K_{MCPP32}$=9200 and V_{max}/K_{mMch4}=18). However, similar to CPP32 and Mch3α, Mch4 is potently inhibited by the DEVD-CHO (SEQ ID NO: 71) peptide (K_{iMch4}=14 nM) and weekly inhibited by Crm A (K_{iMch4}=0.75 μM) (Fernandes-Alnemri et al., supra (1995b)). Since DEVD-CHO (SEQ ID NO: 71) also blocks cell death, this result further indicates that Mch4 is an ASCP which plays a role in the cell death pathway.

Please replace the paragraph (or section) beginning at page 37, line 7, with the following redlined paragraph (or section):

Briefly, Mch5B was subcloned into the mammalian expression vector pcDNA3. The Mch5 cDNA in the vector pBluescript KS was used as a template for PCR amplification of the Mch5 FADD B domain using the following primers: 5' primer: CCTACAGGATCCACTTCTGCCGCATGAGC (SEQ ID NO: 62); 3' primer:

ACTCCTCCCCTTTGCTGAATTCTTAATAGTCGT (SEQ ID NO: 63). The PCR product was cut with BamHI and EcoRI and ligated into BamHI/EcoRI cut pcDNA3 to produce the Mch5/Fadd B/pcDNA3 (MFp) vector. MFp DNA was transduced into DH5α bacteria and DNA was purified. For transfection, MFp or pcDNA3 (1.8 μg) were mixed with the lipofectin reagent (GIBCO Life Technology) and 0.2 μg of plasmid pCMC-SPORT-βgal (GIBCOBRL Catalogue #10586-014) and applied to 50% confluent cultures of MCF7 cells for eight hours at 37°C. The cells were then washed and growth media added. After 36 hours cells were fixed in 10% para-formaldehyde and β galactocidase expression visualized by incubating cells with X-gal substrate solution.

Please delete the section of the application entitled "Sequence Listing" immediately after the section of the specification entitled "Abstract of the Disclosure" on page 39 and insert the enclosed Sequence Listing therefor.